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Serum lipids & lipoprotein profiles of cigarette smokers & passive smokers

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Serum lipids and lipoproteins of 50 active and passive smokers were compared with levels in 25 control subjects. Active smoking resulted in an increase in total cholesterol (T_c) and triglycerides (T_g) as compared to control group. The passive smokers also showed relatively higher levels but the effect was not significant. Active smoking raised the low density lipoprotein cholesterol (LDL_c) and very low density lipoprotein cholesterol ($VLDL_c$) levels whereas high density lipoprotein cholesterol (HDL_c) content was lowered, thus resulting in decreased ratios of HDL_c/T_c and HDL_c/LDL_c . The passive smokers also showed slightly higher levels of LDL_c and $VLDL_c$ but lower levels of HDL_c and a lower HDL_c/LDL_c ratio. Our findings suggest that smoking alters the serum lipids and lipoproteins and these changes are related to the duration and amount of smoking.

Large prospective epidemiological studies have shown strong association between cigarette smoking and several diseases. The potential of developing coronary artery disease in male cigarette smokers is approximately 2.14 times greater than in non-smokers¹. The risk of infarction for both men and women is correlated with the number of cigarettes smoked daily². Association of cigarette smoking, serum lipoproteins and coronary artery disease has been reported by several workers^{3,4}.

Various reports suggest that involuntary inhalation of cigarette smoke by non-smokers causes disease, most notably lung disease^{5,6}. The ubiquitousness of tobacco smoke in homes, work places and public areas makes exposure to environmental tobacco smoke unavoidable^{7,8}.

Asymptomatic non-smokers who are chronically exposed to smoke contaminated air may develop small airway dysfunction^{9,10}. Several studies suggest that passive smoking increases the risk for lung cancer⁶ and aggravates angina pectoris¹¹.

Very little attention has been paid to the effect of passive smoking on serum lipids and lipoproteins. In view of the fact that large population in India is exposed to passive smoking, the present study has been undertaken to know the effect of passive smoking on serum lipids and lipoproteins compared to that in chronic smokers and control subjects.

Material & Methods

A total of 75 subjects of middle income group known to perform moderate physical activity were

included in the present study. Twenty five males who smoked more than 15 cigarettes a day for more than 5 consecutive years were taken as smokers, 25 non-smokers who were chronically exposed to smoke of at least 20 cigarettes a day, in their homes and/or in work places in closed environment of room (s)/ office (s) were taken as passive smokers. Similarly, 25 male non-smokers matched for age and physical activity were taken as control; these individuals lived with strict non-smokers at home and also in their work place.

Subjects suffering from diseases which are known to alter the lipid profile such as diabetes mellitus, uremia, nephrotic syndrome, hypothyroidism, hyperthyroidism and acromegaly, were excluded from the study. Alcoholics and subjects on steroids were also excluded from the study.

Ten ml of blood sample was collected after an overnight fast from each subject. The serum was separated by centrifugation at 3000 rpm for 10 min and was used for lipid and lipoprotein analysis. Total lipids were extracted from the serum¹² and serum total cholesterol (T_c)¹³, serum triglycerides (T_g)¹⁴ in the extracted lipids, HDL_c¹⁵, LDL_c and VLDL_c¹⁶ were estimated. Student's t -test was used for analysis of the data.

Results

Table I shows the comparison of T_c and T_g of smokers and passive smokers with that of control subjects. Significantly higher levels of T_c ($P < 0.05$)

and T_g ($P < 0.01$) were observed in smokers whereas the passive smokers showed marginally higher levels but the values were statistically insignificant when compared to controls. The active smokers had significantly higher levels of LDL_c ($P < 0.01$) and VLDL_c ($P < 0.05$) than controls, whereas these levels of passive smokers showed no significant difference (Table II). The levels of HDL_c in both groups were not significantly different when compared with control. The ratios of HDL_c/ T_c and HDL_c/LDL_c of smokers were significantly lower ($P < 0.01$) as compared to control group. Passive smokers, however, showed significantly lower ($P < 0.05$) ratio of HDL_c/LDL_c only (Table III).

Table IV shows the T_c and T_g of moderate and heavy smokers of different durations. Moderate smokers (smoking 15 to 20 cigarettes/day) who were smoking for more than 15 yr and heavy smokers (smoking more than 20 cigarettes/day) irrespective of the duration showed significantly higher levels of T_c ($P < 0.05$) and T_g ($P < 0.01$) when compared to controls. LDL_c of moderate and heavy smokers were also significantly higher ($P < 0.05$) irrespective of duration of smoking, whereas no significant differences were observed in HDL_c of both groups as compared to control group (Table V). The levels of VLDL_c were also significantly higher in moderate smokers who smoked for more than 15 yr and heavy smokers. Irrespective of the duration of smoking both heavy and moderate smokers had significantly

Table I. Comparison of total cholesterol (T_c) and triglycerides (T_g) of smokers and passive smokers with non-smoker controls

| | T_c (mg/dl) | | T_g (mg/dl) | |
|-----------------------------|---------------|------------------------|---------------|-----------------------|
| | Range | Mean \pm SE | Range | Mean \pm SE |
| Control (n = 25) | 140-270 | 197.72 \pm 7.04 | 80-126 | 100.16 \pm 2.46 |
| Smokers (n = 25) | 180-300 | 233.28 \pm 5.83** | 96-166 | 131.32 \pm 3.77* |
| Passive smokers (n = 25) | 146-266 | 202.00 \pm 4.62 | 80-138 | 106.00 \pm 2.76 |

P values. * < 0.01 ; ** < 0.05 , as compared to controls

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Table II. Comparison of cholesterol of lipoprotein fractions in smokers and passive smokers with controls

| | HDL _c (mg/dl) | | LDL _c (mg/dl) | | VLDL _c (mg/dl) | |
|-----------------------------|--------------------------|---------------------|--------------------------|-----------------------|---------------------------|-----------------------|
| | Range | Mean \pm SE | Range | Mean \pm SE | Range | Mean \pm SE |
| Control (n = 25) | 38-74 | 58.28 \pm 1.80 | 75-174 | 117.68 \pm 5.68 | 17-29 | 21.76 \pm 0.69 |
| Smokers (n = 25) | 40-76 | 52.88 \pm 1.68 | 116-221 | 153.52 \pm 4.96* | 18-37 | 26.88 \pm 1.06** |
| Passive smokers (n = 25) | 40-73 | 55.24 \pm 1.72 | 83-170 | 123.72 \pm 3.81 | 16-32 | 22.80 \pm 0.72 |

P values, * <0.01 ; ** <0.05 , as compared to controlsTable III. Ratio of HDL_c/T_c and HDL_c/LDL_c of smokers and passive smokers compared with controls

| | HDL _c /T _c | | HDL _c /LDL _c | |
|-----------------------------|----------------------------------|----------------------|------------------------------------|-----------------------|
| | Range | Mean \pm SE | Range | Mean \pm SE |
| Control (n = 25) | 0.25-0.36 | 0.30 \pm 0.006 | 0.37-0.68 | 0.51 \pm 0.016 |
| Smokers (n = 25) | 0.17-0.28 | 0.23 \pm 0.006* | 0.24-0.47 | 0.35 \pm 0.012* |
| Passive smokers (n = 25) | 0.18-0.38 | 0.28 \pm 0.008 | 0.24-0.65 | 0.45 \pm 0.016** |

P values, * <0.01 ; ** <0.05 , as compared to controlsTable IV. T_c and T_g of moderate and heavy smokers of different durations

| Duration yr | T _c (mg/dl) | | T _g (mg/dl) | |
|---|------------------------------------|---|------------------------|--|
| | Range | Mean \pm SE | Range | Mean \pm SE |
| Control (n = 25) | 140-270 | 197.72 \pm 7.04 | 80-120 | 100.16 \pm 2.46 |
| Moderate smokers (15-20 cig./day) | < 15 (n = 8) > 15 (n = 6) | 180-246 215.88 \pm 6.92 190-300 243.50 \pm 15.61** | 96-156 120-166 | 117.75 \pm 6.82 136.67 \pm 6.54* |
| Heavy smokers (< 20 cig. day) | < 15 (n = 6) > 15 (n = 5) | 210-266 230.83 \pm 8.09** 217-300 251.80 \pm 13.43** | 118-155 110-156 | 137.50 \pm 6.39* 139.20 \pm 7.81* |

P values, * <0.01 ; ** <0.05 , as compared to controls

higher ($P < 0.01$) HDL_c/LDL_c and HDL_c/T_c ratios as compared to controls (Table VI).

Discussion

There is increasing epidemiological evidence on

the association of cigarette smoking, serum lipoproteins and cardiovascular diseases¹⁷. In the present study, the serum lipids and lipoproteins of cigarette smokers and passive smokers have been compared with strict non-smokers. Comparison of

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Table V. Comparison of cholesterol of lipoprotein fractions of moderate and heavy smokers of different duration with controls

| | Duration, yr | HDL _c (mg/dl) | | LDL _c (mg/dl) | | VLDL _c (mg/dl) | |
|---|-----------------|--------------------------|------------------|--------------------------|----------------------|---------------------------|--------------------|
| | | Range | Mean \pm SE | Range | Mean \pm SE | Range | Mean \pm SE |
| Control (n = 25) | | 38-74 | 58.28 \pm 1.80 | 75-174 | 117.68 \pm 5.68 | 17-29 | 21.76 \pm 0.69 |
| Moderate smokers (15-20 cig./day) | < 15 (n = 8) | 42-64 | 51.00 \pm 2.58 | 120-177 | 141.13 \pm 6.19** | 18-37 | 23.75 \pm 2.08 |
| | > 15 (n = 6) | 48-66 | 59.33 \pm 4.37 | 116-201 | 157.33 \pm 12.94** | 23-33 | 26.83 \pm 1.35** |
| Heavy smokers (< 20 cig./day) | < 15 (n = 6) | 44-58 | 51.50 \pm 2.39 | 134-180 | 151.83 \pm 6.29** | 22-37 | 27.50 \pm 2.17** |
| | > 15 (n = 5) | 40-60 | 49.80 \pm 3.41 | 146-221 | 170.80 \pm 13.33** | 27-36 | 31.20 \pm 1.71* |

P values, * < 0.01; ** < 0.05, as compared to controls

Table VI. Ratio of HDL_c/T_c and HDL_c/LDL_c of moderate and heavy smokers of different durations compared with control

| | Duration, yr | HDL _c /T _c | | HDL _c /LDL _c | |
|---|-----------------|----------------------------------|-------------------|------------------------------------|-------------------|
| | | Range | Mean \pm SE | Range | Mean \pm SE |
| Control (n = 25) | | 0.25-0.36 | 0.30 \pm 0.006 | 0.37-0.68 | 0.51 \pm 0.016 |
| Moderate smokers (15-20 cig./day) | < 15 (n = 8) | 0.18-0.28 | 0.24 \pm 0.014* | 0.25-0.44 | 0.37 \pm 0.021* |
| | > 15 (n = 6) | 0.21-0.28 | 0.24 \pm 0.012* | 0.30-0.47 | 0.38 \pm 0.024* |
| Heavy smokers (> 20 cig./day) | < 15 (n = 6) | 0.20-0.25 | 0.22 \pm 0.008* | 0.29-0.39 | 0.34 \pm 0.040* |
| | > 15 (n = 5) | 0.17-0.24 | 0.20 \pm 0.013* | 0.24-0.38 | 0.30 \pm 0.027* |

P values, * < 0.01, as compared to controls

T_c and T_g levels indicated that cigarette smoking raised T_c and T_g levels. These findings are in accordance with the observations of other workers¹⁸⁻²¹, but contrary to a few who did not observe such an effect²². Since higher T_c and T_g levels are known to be responsible for the development of atherosclerosis²³, it indicates that active cigarette smoking can be a major risk factor for coronary artery disease.

Our findings of raised T_c and T_g levels in moderate smokers who had smoked for more than 15 yr and in heavy smokers (i.e., respective of the duration of smoking) are in agreement with the reports of a number of other workers from different parts of the world^{18, 19, 24}. However, a

few workers did not observe any change in T_c levels in smokers^{20, 25}.

The levels of LDL_c and VLDL_c were higher in heavy smokers or those who had smoked for longer duration. The differences observed in serum lipoproteins between smokers and non-smokers were in accordance with the observations of other workers^{19, 21, 24, 26}. However, Howell²⁷ and Young²⁸ did not observe any differences. The HDL_c of smokers was lower than that of control and passive smokers. Recently, Rastogi *et al.*²⁴ have also reported a similar effect on HDL_c in heavy smokers or those who were smoking for longer duration.

The comparison of the ratios HDL_c/T_c and

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HDL/LDL indicated that smokers had a significantly lower value. Investigators from the Framingham Heart Study²⁸ have proposed that these ratios may be better predictors of coronary risk than T_c or any of the lipoprotein cholesterol levels alone. Lowering of this ratio is known to increase the risk of development of cardiovascular disease. It is thus clear that smoking is a great hazard with respect to cardiovascular diseases. Since the ratio of HDL/LDL is also significantly lower amongst passive smokers, it indicates that not only active smokers but also subjects who are in contact with active smokers are at a relatively higher risk of developing atherosclerosis. The lower degree of risk amongst passive smokers compared to that amongst active smokers could be due to the filtration of smoke in the lungs of the smokers. Some of the components like nicotine and tar are deposited in the lungs of active smokers and therefore the passive smokers are exposed to a lower density of harmful components. Swendsen *et al*²⁹ conducted the multiple risk factor intervention trial to study the effects of passive smoking and the data from their study suggested that passive exposure to cigarette smoke may have a deleterious impact on the health of non-smokers and the non-smokers may be at an increased risk of death through passive exposure to cigarette smoke. Our findings also support this, as the levels of serum lipids and lipoproteins were altered in passive smokers in such a manner that it may have a deleterious effect on cardiovascular system.

The full impact of smoking on cardiovascular disease may not be revealed by available epidemiological surveys as risk ratios derived from these surveys do not necessarily reveal all of the cardiovascular consequences of smoking. It has been demonstrated that heavy smokers are at a higher risk than light smokers³.

In conclusion, our findings suggest that smoking alters the serum lipid and lipoproteins and these changes become more marked with duration and amount of smoking. The passive smokers also show relatively less altered lipid and lipoproteins, in a trend similar to that of smokers. The alteration in the individual value of lipids and

lipoproteins is not significant in case of passive smokers but the results are significant only in case of ratios of HDL/ T_c and HDL/LDL. As decrease in this ratio is responsible for the development of atherosclerosis, the results indicate that even the passive smokers are at a relatively higher risk of developing coronary heart disease.

References

1. Shapiro, S., Weinblatt, E., Frank, C.W. and Sagar, R.V. Incidence of coronary heart disease in a population insured for medical care (HIP): myocardial infarction, angina pectoris and possible myocardial infarction. *Am J Public Health* 59 Suppl (1969) 1.
2. Kaufman, D.W., Helmrich, S.P., Rosenberg, L., Miettinen, O.S. and Shapiro, S. Nicotine and carbon monoxide content of cigarette smoke and the risk of myocardial infarction in young men. *N Engl J Med* 308 (1983) 409.
3. Brischetto, C.S., Connor, W.E., Connor, S.L. and Matarazzo, J.D. Plasma lipid and lipoprotein profiles of cigarette smokers from randomly selected families: enhancement of hyperlipidemia and depression of high density lipoprotein. *Am J Cardiol* 52 (1983) 675.
4. Stamler, J. Primary prevention of coronary heart disease. The last 20 years. *Am J Cardiol* 47 (1981) 722.
5. Akiba, S., Kato, H. and Blot, W.J. Passive smoking and lung cancer among Japanese women. *Cancer Res* 46 (1986) 4804.
6. Dimitrios, T., Kalandidi, A., Sparros, L. and MacMahon, B. Lung cancer and passive smoking. *Int J Cancer* 27 (1981) 1.
7. Tager, I.B., Weiss, S.T., Roemer, B. and Speizer, F.E. Effect of parental cigarette smoking on the pulmonary function of children. *Am J Epidemiol* 110 (1979) 15.
8. Weiss, S.T. Passive smoking and lung cancer: What is the risk? *Am Rev Respir Dis* 133 (1986) 1.
9. Kauffmann, F., Tessier, J.F. and Oriol, P. Adult passive smoking in the home environment: a risk factor for chronic airflow limitation. *Am J Epidemiol* 117 (1983) 269.
10. White, J.R. and Froeb, H.F. Small-airways dysfunction in nonsmokers chronically exposed to tobacco smoke. *N Engl J Med* 302 (1980) 720.
11. Aronow, W.S. Effect of passive smoking on angina pectoris. *N Engl J Med* 299 (1978) 21.
12. Folch, J., Lees, M. and Sloane-Stanley, G.H. A simple method for the isolation and purification of total lipids from animal tissues. *J Biol Chem* 226 (1957) 497.

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13. Zlatkis, A., Zak, B. and Boyle, A.J. A new method for the direct determination of serum cholesterol. *J Lab Clin Med* 41 (1953) 486.
14. Van Handel, E. and Zilversmit, D.B. Micromethod for the direct determination of serum triglycerides. *J Lab Clin Med* 50 (1957) 152.
15. Lopes-Virella, M.F., Stone, P., Ellis, S. and Colwell, J.A. Cholesterol determination in high density lipoproteins separated by three different methods. *Clin Chem* 23 (1977) 882.
16. Wilson, D.E. and Spiger, M.J. A dual precipitation method for quantitative plasma lipoprotein measurements without ultracentrifugation. *J Lab Clin Med* 82 (1973) 473.
17. Grundy, S.M., Greenland, P., Herd, A., Huebsch, J.A., Jones, R.J., Mitchell, J.H. and Schlent, R.C. Cardiovascular and risk factor evaluation of healthy American adults. *Circulation* 75 (1987) 1340A.
18. Balawa, V.S., Gupta, M.C., Maheshwari, V.D. and Bhansali, A. Effect of prolonged smoking and alcohol on lipid profile, separately and in combination. *J Assoc Physicians India* 31 (1983) 573.
19. Freedman, D.S., Srinivasan, S.R., Shear, C.L., Hunter, S.M., Croft, J.B., Webber, L.S. and Berenson, G.S. Cigarette smoking initiation and longitudinal changes in serum lipids and lipoproteins in early adulthood: the Bogalusa heart study. *Am J Epidemiol* 124 (1986) 207.
20. Garg, J.P., Gupta, R.S., Agarwal, M.P. and Bhandari, V.M. Effect of smoking on serum lipids and lipoproteins in healthy subjects and patients of old myocardial infarction and hypertension. *Indian J Med Sci* 37 (1983) 63.
21. Richmond, W., Seviour, P.W., Tsai, T.K. and Elkeles, R.S. Impaired intravascular lipolysis with changes in concentrations of high density lipoprotein sub-classes in young smokers. *Br Med J* 295 (1987) 246.
22. Kontinen, A. and Rajasalmi, M. Effect of heavy cigarette smoking on postprandial triglycerides, free fatty acids and cholesterol. *Br Med J* 1 (1963) 850.
23. Keys, A. Coronary heart disease in seven countries. *Circulation* 41 Suppl (1970) 1.
24. Billimoria, J.D., Pozner, H., Metelaar, B., Best, F.W. and James, D.C. Effect of cigarette smoking on lipids, lipoproteins, blood coagulation, fibrinolysis and cellular components of human blood. *Atherosclerosis* 21 (1975) 61.
25. Yeung, D.L. Relationship between cigarette smoking, oral contraceptives and plasma vitamins A, E, C and plasma triglycerides and cholesterol. *Am J Clin Nutr* 29 (1976) 1216.
26. Rastogi, R., Shrivastava, S.S.L., Mehrotra, T.N., Singh, V.S. and Gupta, M.K. Lipid profile in smokers. *J Assoc Physicians India* 37 (1989) 764.
27. Howell, R.W. Smoking habits and laboratory tests. *Lancet* ii (1970) 152.
28. Wilson, P.W., Garrison, R.J., Castelli, W.P., Fainleib, M., McNamara, P.M. and Kannel, W.B. Prevalence of coronary heart disease in the Framingham offspring study: role of lipoprotein cholesterol. *Am J Cardiol* 46 (1980) 649.
29. Svendsen, K.H., Kuller, L.H., Martin, M.J. and Ockene, J.K. Effects of passive smoking in the multiple risk factor intervention trial. *Am J Epidemiol* 126 (1987) 783.

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